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EXAMINER	
MCGARRY, SEAN	
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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Paper No. 12

Application Number: 09/490,187
Filing Date: 1/23/00
Appellant(s): Chaudhary

Richard Aron Osman
For Appellant

EXAMINER'S ANSWER

This is in response to appellant's brief on appeal filed 8/30/01.

(1) Real Party in Interest

A statement identifying the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

A statement identifying the related appeals and interferences which will directly affect or be directly affected by or have a bearing on the decision in the pending appeal is contained in the brief.

(3) Status of Claims

The statement of the status of the claims contained in the brief is correct.

(4) Status of Amendments After Final

No amendment after final has been filed.

(5) Summary of Invention

The summary of invention contained in the brief is correct.

(6) Issues

The appellant's statement of the issues in the brief is correct.

(7) Grouping of Claims

The appellant's statement in the brief that certain claims do not stand or fall together is not agreed with because the claims are all rejected under the same grounds and appellant has not provided any reasons why the claims should stand separately.

The copy of the appealed claims contained in the Appendix to the brief is correct.

(9) Prior Art of Record

No prior art is relied upon by the examiner in the rejection of the claims under appeal.

(10) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claims 1-21 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The instant invention is drawn to methods of detecting the presence of or predisposition to an ectodermal disorder via a "TAJ" gene or gene product and to methods of treating "TAJ" associated ectodermal disorders.

The instant specification discloses a "TAJ" nucleic acid and protein sequence (SEQ ID NO: 1 and 2). The specification discloses at page 1 that there are over 150 different ectodermal dysplasia syndromes. The specification discloses at page 3 that ectodermal disorders may arise from temporal, developmental, quantitative or qualitative TAJ misexpression and that a wide variety of causalities may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function, etc. Table 1 of the instant specification discloses "TAJ" mutants that result in truncated TAJ proteins. This table does not provide any indication what ectodermal dysplasia might be associated with the disclosed mutants and further does not provide any guidance as to how or where these sequences were detected or constructed and are all based on SEQ ID NO: 1 and 2. Further it does not tell one whether these specific mutants are associated with autosomal dominant or recessive ectodermal dysplasia in hetero or homozygous mutants. The instant specification asserts at page 3 that detection can be made directly (detecting protein or nucleic acid), indirectly (detecting specific function of the target) or inferentially (detecting a diagnostic sequence in a genomic or proteomic database). Table 2 discloses "exemplary allele specific TAJ antibodies and hybridization probes". Table 2 indicates specific binding and specific hybridization as "+++". There is no legend that defines

what "+++" indicates. Does "+++" represent that there is high level, low level, intermediate binding and relative to what? Table 3 discloses "exemplary agents shown to allele specifically modulate functional expression of a TAJ gene or gene product". Table 3 uses "+++" to designate modulation. Does "+++" mean increased activity , decreased activity, what level and relative to what? The specification discloses in Example I the differential expression of murine TAJ in mouse embryos and provides a prophetic animal model of Cloustron syndrome. Example II discloses that TAJ activates JNK upon transient over expression of TAJ and discloses a cJun transcriptional activation assay with no results. Example III is a protocol for high throughput TAJ polypeptide-Traf binding interference assay with no results. Example IV appears to be a prophetic example of genomic diagnosis of suspected Cloustron's syndrome. Example V (page 12) is a prophetic example of corrective gene transfer in ectodermal dysplasia. Second example V (page 15) is a prophetic example of localized in vivo genotypic and phenotypic correction.

The instant specification does not provide sufficient guidance or examples that would show by correlation the practice of the instant invention without undue experimentation. Since there are so many (over 150) disease states and little guidance for one of skill in the art to detect or treat such diseases based on the instant specification one would be left to undue trial and error experimentation. For example, the instant invention is based on the association of a TAJ gene and ectodermal dysplasia. The instant specification is sketchy as to the correlation of TAJ and ectodermal dysplasia. No specific examples or discussion is provided that teaches one of skill in

the art the role TAJ genes or gene products in and ectodermal dysplastic state. The specification provides tables that appear to be ambiguous in what they disclose. These tables nor the specification provides any guidance as to what ectodermal dysplasia diseases even the mutants of Table 1 are associated. The instant invention appears to be an invitation for one in the art to draw correlations to any nucleic acid sequence or protein that might be a TAJ to the numerous disease states contemplated. This is not a simple task considering the large number of diseases that manifest in numerous different ways in different cells and involve different biological pathways. For example, ectodermal disorders may arise from temporal, developmental, quantitative or qualitative TAJ misexpression and that a wide variety of causalities may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function, etc and one of skill in the art is left to make these correlations themselves. Claim 1 even recites that a correlation must be made. The instant specification has essentially demonstrated that SEQ ID NO:2 activates the JNK pathway upon over expression and that hTAJ is expressed in prostate cell and in fetal kidney cells (fetal kidney cell line) and that TAJ is differentially expressed in murine fetuses. The information provided in the Tables is unclear as to how it provides evidence for TAJ association in ectodermal dysplasia. Without this basic knowledge or correlative evidence or guidance it is unclear how one of skill in the art could practice the claimed invention without undue trial and error experimentation.

(11) Response to Argument

Appellant argues that the specification thoroughly teaches and exemplifies the method steps required to practice the claimed methods including detection methodologies and correlating methodologies. Appellant refers to page 9 in regard to in situ and chromosomal hybridization. At this location it is disclosed that the TAJ gene was localized to a chromosomal region linked to a marker for Cloustron syndrome. This example does not provide any evidence that the TAJ is involved in the manifestation of Cloustron syndrome but shows that the gene is located near the locus associated with Cloustron syndrome, for example. The specification does not provide even this preliminary data for any of the other over 150 know ectodermal disorders. The specification asserts, for example, that ectodermal disorders may arise from temporal, developmental, quantitative or qualitative TAJ misexpression and that a wide variety of causalities may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function, etc., and one of skill in the art is left to make these correlations themselves for the over 150 know ectodermal disorders. The specification provides no specific guidance for one in the art to make these correlations. Appellant asserts that one in the art has to do no more than cross reference known clinical correlates. The instant specification does not provide of such a correlates to any specific TAJ mutant and any specific ectodermal disorder. Appellant asserts that one may simply refer to

known correlates. No such known correlates are provided or cited in the instant specification. Appellant asserts that the specification provides exemplification of an animal model. This prophetic animal model is directed to a Cloustron syndrome model and no guidance is provided, for example, for the other over 150 know ectodermal disorders. Appellant also asserts that the specification provides exemplification of a clinical diagnosis. This prophetic example too is drawn specifically to Cloustron syndrome and provides no guidance for the other over 150 know ectodermal disorders.

Appellants argues the "criticism" of the data Tables in the specification. Appellant argues the meaning of the data in the filed Brief and asserts the meaning of the "+++" designation in these Tables. The asserted meanings for "+++" can not be found in the specification as filed.

Appellant further argues that all that is required by the instant methods is that one correlate be found and asserts that such a correlation is exemplified in Example IV. Example IV is drawn specifically to Clouston syndrome. The scope of the claims is drawn to any and all ectodermal dysplasias as appellants summary of the invention appears to indicate, for example. It is unclear how Example IV provides guidance for the over 150 different ectodermal dysplasias that manifest in numerous different ways in different cells and involve different biological pathways. The specification asserts, for example, that ectodermal disorders may arise from temporal, developmental, quantitative or qualitative TAJ misexpression and that a wide variety of causalities may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products

which may in turn regulate TAJ expression or TAJ function. This assertion of the specification does not take into account the numerous other genes that may de directly and indirectly involved in the manifestation of ectodermal dysplasias. For example, the specification asserts that the human homolog of Mouse *dl* have been reported to cause autosomal recessive and dominant hypohidrotic ectodermal dysplasia. The instant specification does not indicate how the TAJ gene is related to human homolog of the mouse dl gene or how such an observation correlates to the instant invention. One in the art is left to determine the temporal, developmental, quantitative or qualitative TAJ misexpression and the wide variety of causalities that may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function that may be involved in any specific ectodermal dysplasia and determine if, for example, any of the above indicate a predisposition to a specific ectodermal dysplasia or indicates that one is suffering from a specific ectodermal dysplasia and further devise a course of treatment based on the work performed outside the disclosure of the instant specification.

Appellant argues that the treatment of ectodermal dysplasia has been thoroughly taught in the instant specification. This is not agreed with, since, for example, one in the art has not been provided with the most basic information, such as how is TAJ specifically involved in any particular disease. Is any observed variance in TAJ expression the cause of, or an effect of, or perhaps linked to an ectodermal dysplasia? One in the art would be required to make such

determination by undue trial and error experimentation. For example the specification asserts that ectodermal disorders may arise from temporal, developmental, quantitative or qualitative TAJ misexpression and that a wide variety of causalities may effect such misexpression such as genetic lesions or mutations gene itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function. One in the art is left to make such determinations for any particular ectodermal dysplasia to determine if one is suffering from, is predisposed to or to treat any particular ectodermal dysplasia.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

Sean McGarry
February 5, 2002

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